TITLE

Increased Interleukin-1 Signaling and Reactive Astrogliosis Following Blast-induced Traumatic Brain Injury

ABSTRACT

Traumatic Brain Injury (TBI) affects nearly 2.8 million people in the United States annually, resulting in nearly 250,000 hospitalizations, 50,000 deaths, and an estimated annual cost of 70 billion dollars. Currently there are no FDA approved medications to combat the chronic effects of TBI. Those affected offer suffer from chronic neuropsychiatric disorders such as depression and anxiety. Neuroinflammation is characteristic of brain trauma and results in the production of pro-inflammatory cytokines such as interleukin-1α (IL-1α) and interleukin-1β (IL-1β). We hypothesize that the acute inflammatory signaling involving interleukin-1 (IL-1) post TBI drives the behavioral alterations related to such neuropsychiatric disorders. To test this hypothesis, adult C57Bl/6J mice underwent TBI or sham (control) treatments using a murine model of blast-induced TBI. Acute TBI resulted in increased righting reflex times, decreases in locomotor activity and increases in despair like behavior. Further, an increase in cortical and hippocampal *il-1α*, *il-1β* and IL-1 receptor *(il-1r1)* mRNA expression was observed in comparison to sham mice. As well, TBI resulted in increased mRNA expression of the astrocytic marker glial fibrillary acidic protein (GFAP) as compared to sham animals’ post-injury, indicative of reactive astrogliosis. Future studies are aimed at understanding the role of IL-1 signaling in the central nervous system (CNS) using IL-1R1 knockout mice. This research may provide information about potential drug targets to treat those with TBI.